

The validity of these reservations was clearly demonstrated by Benson in a recent study of about 25,000 patients in which the value of stethoscopic evaluation of FHR was correlated with infant follow-up. He found that auscultatory evaluation of FHR had little or no value for the early detection of fetal distress and was useful only in the most extreme circumstances.

With the introduction of electronic techniques for continuous monitoring of the FHR and concomitant fetal biochemical studies, a better understanding of FHR has emerged. It is now clear that the FHR changes *with* contractions are much more important than those taking place in the interval *between* contractions and an assessment of the FHR *pattern* yields much more information than stethoscopic evaluation of FHR *levels*.

It has also become apparent that the most common cause of fetal distress during labor and delivery is due to umbilical cord compression and that continuous monitoring of the FHR is a very efficient method for the early detection of umbilical cord compression. This complication of labor and delivery is not limited to the high-risk pregnancy, but may also become suddenly manifest in the patient who has had a normal antepartum course. There is a need, therefore, for a technique for continuously monitoring the FHR during labor and delivery. Currently, the most widely used techniques employ an electrode which is attached directly to the fetal presenting part to record the fetal electrocardiogram from which the beat-to-beat FHR is computed. In addition a catheter is introduced transcervically to record the uterine activity.

While this technique is valuable for the evaluation of patients where the membranes are ruptured and the cervix is 2 cm dilated, it is not applicable in all situations where fetal monitoring may be desirable. Fortunately techniques have been devised also to record both FHR and uterine activity from the maternal abdominal wall. While the data is not as precise as that obtained by direct techniques, it is more than adequate for screening purposes. If problems are uncovered with this screening technique, direct techniques may then be employed.

Fetal monitoring using direct techniques is practical and can be used by all practicing obstetricians, regardless of the size of the hospital, and it should be used for the monitoring of all

high-risk patients. Ideally, the FHR of all patients in labor should be checked continuously. The cost of the equipment makes this impossible at present but equipment for this purpose is being developed.

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REFERENCES

- Benson RC, Shubeck F, Deutschberger J, et al: Fetal heart rate as a predictor of fetal distress: A report from the collaborative project. *Obstet Gynec* 32:259, 1968
Hon EH: *An Atlas of Fetal Heart Rate Patterns*. New Haven, Harty Press Inc., 1968

Clinical Significance of Folic Acid Metabolism in Pregnancy

Folic acid deficiency in its various forms is more common than was previously recognized. Folates, a generic term used to include all pteroylglutamic acids, are involved in DNA, RNA and protein biosynthesis acting as coenzymes for one-carbon transfers necessary for the formation of their purine, pyrimidine and amino acid precursors. The most overt effect of folic acid deficiency in pregnancy is on the bone marrow, where it results in megaloblastic anemia of pregnancy. Lesser degrees of folic acid deficiency may be present without overt anemia. The true incidence in the United States of folate deficiency in pregnancy with or without megaloblastic anemia is unknown. That deficiency is, however, by far the most common of all water-soluble vitamin deficiencies in this country. Pregnancy demands for folate are unusually great. Whereas normal women who are not pregnant require about 50 micrograms a day, women who are pregnant in the last trimester have a total requirement of from 350 to 500 micrograms a day. The presence of multiple fetuses and placentas or coexisting hemolytic anemia causes further demands for folate during pregnancy. However, all patients with overt megaloblastic anemia, including those pregnant with twins and with hemolytic anemia, respond adequately to 1 mg a day of folic acid even while receiving a low folate diet.

There is an increasing body of evidence that the many complications of pregnancy that have

been thought to be associated with folic acid deficiency probably bear no relationship to deficiency of this water-soluble vitamin. Careful investigation from several centers has shown no relationship between such deficiency and placental abruption, toxemia of pregnancy or fetal malformation. It can be concluded that although folate does not appear to play a role in these pregnancy complications, megaloblastic anemia due to folate deficiency is not uncommon. There-

fore, if a supplemental medication other than iron is to be given in pregnancy, it should certainly include folic acid containing from 500 to 1,000 micrograms of folate.

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REFERENCES

- Herbert V: Aseptic addition method for lactobacillus casei assay of folate activity in human serum. *J Clin Path* 19:12-16, 1966
 Scott DE, Whalley PJ, Pritchard JA: Maternal folate deficiency and pregnancy wastage II. Fetal malformation. *Obstet and Gynec* 36:26-28, 1970

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